

**ON THE GLOBAL INCREASE OF HARMFUL ALGAL BLOOMS.** *Memoirs of the Queensland Museum* 343(3): 560. 1994:— Harmful algal blooms have occurred throughout recorded history but during the past two decades they have increased in frequency, intensity, and geographic distribution; their effects on human health and economics have increased accordingly. To some extent, this reflects our increased awareness of toxic species and the enormous expansion in aquaculture efforts. Evidence is accumulating, however, that human activities contribute significantly to this increase through the stimulation of exceptional blooms by cultural eutrophication (e.g. from domestic, industrial and agricultural

wastes; acid precipitation, deforestation and increased runoff from cleared land) and by the spreading of nuisance organisms in ships' ballast water. The global distribution of these phenomena is illustrated with examples from Japan, North America, Europe, South-East Asia and Australia, and involving dinoflagellates, diatoms, prymnesiophytes, raphidophytes and cyanobacteria.

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**CIGUATOXIN-1 INDUCES SPONTANEOUS SYNAPTIC ACTIVITY IN ISOLATED SYMPATHETIC GANGLIA OF GUINEA PIGS** *Memoirs of the Queensland Museum* 34(3): 560. 1994:— An electrophysiological study has been undertaken of the actions of purified ciguatoxin-1 (CTX-1) on the neurones of guinea pig sympathetic ganglia isolated in vitro, using conventional intracellular microelectrode techniques. Low concentrations of CTX-1 (0.2-0.8nM) applied even briefly (<15min) via the perfusing solution induced a dramatic increase in the spontaneous occurrence of excitatory synaptic potentials (ESPs) which persisted for many hours. The amount and pattern of activity varied between neurones and occurred in the absence of any change in passive or active electrical properties of the neurones themselves. Single supramaximal preganglionic stimuli evoked a summed response which was unaltered after exposure to CTX-1, but

was followed by a variable duration high frequency burst of ESPs. These bursts resembled those occurring spontaneously in the same cell, and apparently arose from individual preganglionic axons. The effects were abolished by reduced  $Ca^{2+}$ ,  $\omega$ -conotoxin, low doses of TTX or raised divalent cation concentrations. The results indicate that some preganglionic axons have CTX-binding sites that open  $Na^+$  channels causing spontaneous depolarization and initiating repetitive discharges.

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